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Maintained vasodilatory response to cromakalim after inhibition of nitric oxide synthesis.

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Activation of vascular smooth-muscle adenosine triphosphate-sensitive potassium channels (KATP channels) causes membrane hyperpolarization, reduced entry of Ca2+ through L-type voltage-gated Ca2+ channels, and subsequent smooth-muscle relaxation. Conversely, opening of endothelial KATP channels elicits hyperpolarization but may induce Ca2+ influx and stimulation of endothelium-derived nitric oxide (EDNO) because these cells appear not to possess L-type Ca2+ channels. We therefore hypothesized that EDNO contributes to KATP channel-mediated vasodilation. To test this hypothesis, we examined vasodilatory responses to the KATP channel opener cromakalim in conscious rats, perfused rat tail artery segments, and isolated perfused rat lungs in the presence or absence of the EDNO synthesis inhibitor Nomega-nitro-L-arginine (L-NNA). Additionally, we compared the effect of cromakalim with the EDNO-dependent dilator bradykinin on NO production and intracellular Ca2+ in cultured rat pulmonary artery endothelial cells. Vasodilatory profiles to cromakalim were unaffected by L-NNA in conscious rats, tail arteries, and isolated lungs. Consistent with these results, cromakalim had no apparent effect on either NO synthesis or Ca2+ levels in cultured endothelial cells. These data suggest a lack of a role for EDNO in contributing to KATP-channel-mediated vasodilation in the rat.

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